THE INFLUENCE OF FEVER ON THE PAINS OF LOCOMOTOR ATAXIA.

By Charles W. Burr, M.D., of Philadelphia,
professor of mental diseases in the university of pennsylvania.

I know of no observations concerning the effect of intercurrent febrile diseases upon the pains occurring in locomotor ataxia. My own data are not extensive enough to use to draw positive conclusions as to whether any febrile disease always influences these pains, either by increasing or diminishing them, nor to explain the process by which the fever acts. The subject is, however, of enough importance to merit consideration. My attention was first called to the matter by the great increase in the severity of the pains of an ataxic during an intercurrent attack of malarial fever. The patient was syphilitic and alcoholic. His illness had begun several years before his admission to the Philadelphia Hospital with not very severe shooting pains confined to the legs, followed after many months by a staggering gait. When examined he presented marked ataxia of motion and station, absent knee-jerks, Argyll-Robertson pupils, Charcot joints in either knee, and areas of anesthesia in either leg. He did not give a history of, nor exhibit during his stay in the hospital, gastric or other crises. In May, 1897, he was suddenly seized with a chill, followed by fever, sweat, and a fall of temperature to normal. The attacks occurred daily for five days, the temperature rising every afternoon to 104 F. and falling a few hours later to normal. From the beginning of each chill until the temperature began to fall he complained bitterly of violent lancinating pains in the legs. At no other time during his stay in the hospital, a period of many months, did he exhibit any such evidence of pain. He himself said that he had never felt such. On the fourth day his blood was examined and the malarial organism found. Quinine was given in rather large doses, and after the fifth day the chills ceased, fever did not return, and the severe pains stopped. Several years later another patient at the Philadelphia Hospital underwent a similar experience. His also was a clear-cut case of locomotor ataxia. He had been in the hos-

pital several weeks when he suddenly was seized with a chill with fever. He had three chills on alternate days, the temperature rising during two to 105 F. and in one to 104 F. During each attack, from the beginning of the chill until there was a distinct fall in the temperature, he complained of great pain shooting through the arms and legs. (In this man the ordinary ataxic pains, which were not severe, involved not only the legs, but the arms also.) On the sixth day Dr. C. Y. White found malarial organisms in the blood. Quinine was given, the chills and fever ceased, and the pains resumed their usual character.

About a year ago a gentleman who had had locomotor ataxia for some years, and in whom the disease had progressed very slowly, but who always suffered greatly for some hours before a storm, and only then from pains in the legs and left intercostal muscles, and who usually was relieved as soon as the storm broke, gave himself a deep hypodermic injection of morphia. He was relieved, but two days later fever appeared and with it terrific pain. At first I was at a loss to discover the cause of the fever. The temperature chart was that of acute pus poisoning. In a few days he told me of the hypodermic injection, and said there was a very sore and sensitive spot on one thigh. On examination a large abscess burrowing deeply was found. A surgeon opened it, poulticed it, and after two weeks the wound healed and the fever ceased. During the entire time that the fever lasted, not only when the temperature was high, but also in the intermissions, the patient suffered excruciating lancinating pains in the legs and left intercostal muscles. Never before or since has he so suffered for so long a time. As a rule his attacks lasted only a few hours. I believe, therefore, that the increase of pain was caused by the septicemia.

Another case was that of an old woman whose cord disease was complicated by epithelioma of the cervix, vagina and bladder, the latter arising several years after her admission to the hospital. For six or seven years she had had marked but not severe ataxic pain. The ataxia was so great that she could not walk or even stand, nor could she feed herself. Argyll-Robertson pupil was present, and there was gray degeneration of both optic nerves. The knee-jerks were abolished. She had no gastric nor other crises Sensibility to touch was preserved in

the hands. Some months before her death she began to complain of difficulty in micturition, which at first was thought to be due to her spinal disease, then of uterine symptoms and later to emaciate. Not long after an irregular see-saw fever developed. The temperature ranged irregularly from 97 F. to 105 F. She had irregularly recurring chills, and during each chill complained bitterly of shooting pains. Her blood showed nothing. The only lesions found at necropsy other than the posterior sclerosis and the epithelioma were chronic interstitial nephritis and an area of old healed tuberculosis in one lung. The fever evidently was septic and secondary to the malignant disease.

These are the only cases I can recall, and of which I have notes, in which fever from any cause has been associated with an increase of pain. I have seen quite a large number of patients die from febrile affections accompanied with chills, in whom the fever was not associated with pain; but they were all, so far as I remember, people in whom pain had ceased to be a symptom years before. In such circumstances fever would not be expected to cause pain. Fever in general is surely not provocative of tabetic pains; at least my personal experience is against such an opinion. I have studied quite a number of patients who, while still subject to pain, developed pulmonary phthisis or cystitis, with a consequent pyelo-nephritis, and in none was there any increase in the severity of the characteristic pain. In two cases of facial erysipelas with quite marked fever there was no pain. It would seem as if only in those diseases in which chills are associated with fever is there any increase in the ataxic pain. It scarcely needs to be mentioned that fever is not a symptom of locomotor ataxia. Dr. W. H. Riley (JOURNAL OF NERVOUS AND MENTAL DISEASE, Sept., 1898), however, found a rise in the temperature during paroxysms of pain in six out of sixty-one cases. He gives neither the cause nor the height of the fever. Bramwell, on the other hand (*Brain*, Part I., 1902) in a study of the temperature of twenty-five cases over a period of twelve days, found fever absent in all except one, who was suffering from influenza. It is not stated whether any of these had paroxysms of pain during the investigation. Two writers, Pell and Oppler, regard "febrile crises" as part of the symptomatology of locomotor ataxia. P. K. Pell (*Berliner klin. Wochenschr.*, June 26, 1896)

reported a case in which the attacks were characterized by great pain in the extremities, high fever, and vomiting lasting one day, chill, and sweat followed by herpes labialis. In one attack the eyes were red and inflamed. He regarded the attacks as being similar to gastric and other crises and offered in explanation of their causation periodic discharge of certain nerve centers in consequence of chemical irritation. In 1902 Dr. Bruno Oppler (*Berliner klin. Wochenschr.*, April 14th) accepted the reality of the occurrence of febrile crises and reported a case in which it was claimed that gastric crises were replaced by them. The attacks were characterized by weakness and fever lasting one day and recurring every six or eight weeks. They were not associated with herpes of the lips. Malarial fever was excluded. Oppler was undecided whether the crises were due to central irritation of the temperature centers or to absorption of toxic matters caused by the suppression of the gastric crises. These are the only papers on the subject I have been able to find, and it would seem that on account of the rarity of the condition it would be justifiable to regard it as a complication rather than as a symptom of locomotor ataxia. Herpes labialis occurs in locomotor ataxia occasionally, but not with sufficient frequency to cause it to be regarded as part of the disease.